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<input type="checkbox"/>	L4	L3 and (kidney or fibrosis or glomerulonephritis or nephropathy or lupus)	16
<input type="checkbox"/>	L3	(zveg4 or pdgf adj d)	21

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=> s (zveg4 or pdgf(w)d)  
L1 174 (ZVEGF4 OR PDGF(W) D)

=> s l1 and antibod?  
L2 37 L1 AND ANTIBOD?

=> s l2 and (kidney or fibrosis or mesangia? or nephritis or lupus)  
L3 20 L2 AND (KIDNEY OR FIBROSIS OR MESANGIA? OR NEPHRITIS OR LUPUS)

=> dup rem l3  
PROCESSING COMPLETED FOR L3  
L4 10 DUP REM L3 (10 DUPLICATES REMOVED)

=> dis ibib abs l4 1-10

L4 ANSWER 1 OF 10 MEDLINE on STN DUPLICATE 1  
ACCESSION NUMBER: 2006170498 IN-PROCESS  
DOCUMENT NUMBER: PubMed ID: 16510766  
TITLE: Antagonism of PDGF-D by human  
antibody CR002 prevents renal scarring in  
experimental glomerulonephritis.  
AUTHOR: Ostendorf Tammo; Rong Song; Boor Peter; Wiedemann Stefanie;  
Kunter Uta; Haubold Ulrike; van Roeyen Claudia R C; Eitner  
Frank; Kawachi Hiroshi; Starling Gary; Alvarez Enrique;  
Smithson Glennda; Floege Jurgen  
CORPORATE SOURCE: Division of Nephrology, University Hospital Aachen,  
Pauwelsstrasse 30, D-52074 Aachen, Germany..  
tostendorf@ukaachen.de  
SOURCE: Journal of the American Society of Nephrology : JASN, (2006  
Apr) Vol. 17, No. 4, pp. 1054-62. Electronic Publication:  
2006-03-01.  
Journal code: 9013836. ISSN: 1046-6673.  
PUB. COUNTRY: United States  
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)  
LANGUAGE: English  
FILE SEGMENT: NONMEDLINE; IN-PROCESS; NONINDEXED; Priority Journals  
ENTRY DATE: Entered STN: 28 Mar 2006  
Last Updated on STN: 15 Apr 2006  
AB Glomerular mesangial cell proliferation and/or matrix  
accumulation characterizes many progressive renal diseases. PDGF  
-D was identified recently as a novel mediator of  
mesangial cell proliferation in vitro and in vivo. This study  
investigated the long-term consequences of PDGF-D  
inhibition in vivo. Rats with progressive mesangioproliferative  
glomerulonephritis (uninephrectomy plus anti-Thy-1.1 antibody)  
received the PDGF-D-neutralizing, fully human mAb  
CR002 on days 3, 10, and 17 after disease induction. Glomerular  
mesangioproliferative changes on day 10 were significantly reduced by  
anti-PDGF-D treatment as compared with control  
antibody. Eight weeks after disease induction, anti-PDGF

-D therapy significantly ameliorated focal segmental glomerulosclerosis, podocyte damage (de novo desmin expression), tubulointerstitial damage, and fibrosis as well as the accumulation of renal interstitial matrix including type III collagen and fibronectin. Treatment with anti-PDGF-D also reduced the cortical infiltration of monocytes/macrophages on day 56, possibly related to lower renal cortical complement activation (C5b-9 deposition) and/or reduced epithelial-to-mesenchymal transition (preserved cortical expression of E-cadherin and reduced expression of vimentin and alpha-smooth muscle actin). In conclusion, these data provide evidence for a causal role of PDGF-D in the pathogenesis of renal scarring and point to a new therapeutic approach to progressive mesangioproliferative renal disease.

L4 ANSWER 2 OF 10 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN  
 ACCESSION NUMBER: 2003:519624 BIOSIS  
 DOCUMENT NUMBER: PREV200300522712  
 TITLE: Method of treating fibroproliferative disorders.  
 AUTHOR(S): Hart, Charles E. [Inventor, Reprint Author]; Topouzis, Stavros [Inventor]; Gilbertson, Debra G. [Inventor]  
 CORPORATE SOURCE: Seattle, WA, USA  
 ASSIGNEE: ZymoGenetics, Inc.  
 PATENT INFORMATION: US 6630142 20031007  
 SOURCE: Official Gazette of the United States Patent and Trademark Office Patents, (Oct 7 2003) Vol. 1275, No. 1.  
<http://www.uspto.gov/web/menu/patdata.html>. e-file.  
 ISSN: 0098-1133 (ISSN print).  
 DOCUMENT TYPE: Patent  
 LANGUAGE: English  
 ENTRY DATE: Entered STN: 5 Nov 2003  
 Last Updated on STN: 5 Nov 2003

AB Materials and methods for reducing cell proliferation or extracellular matrix production in a mammal are disclosed. The methods comprise administering to a mammal a composition comprising a therapeutically effective amount of a zveg4 antagonist in combination with a pharmaceutically acceptable delivery vehicle. Exemplary zveg4 antagonists include anti-zveg4 antibodies, inhibitory polynucleotides, inhibitors of zveg4 activation, and mitogenically inactive, receptor-binding variants of zveg4. The materials and methods are useful in the treatment of, inter alia, fibroproliferative disorders of the kidney, liver, and bone.

L4 ANSWER 3 OF 10 CAPLUS COPYRIGHT 2006 ACS on STN  
 ACCESSION NUMBER: 2003:930854 CAPLUS  
 DOCUMENT NUMBER: 140:1645  
 TITLE: Cloning and characterization of a new family of human secreted proteins - SCUBE1, SCUBE2 and SCUBE3 differentially expressed in vascular endothelium, and therapeutic use thereof  
 INVENTOR(S): Yang, Ruey-bing; Ng, Chi Kin Domingos; Tomlinson, James E.; Komuves, Laszlo G.; Topper, James N.; Robison, Keith E.  
 PATENT ASSIGNEE(S): Millennium Pharmaceuticals, Inc., USA  
 SOURCE: U.S. Pat. Appl. Publ., 86 pp.  
 CODEN: USXXCO  
 DOCUMENT TYPE: Patent  
 LANGUAGE: English  
 FAMILY ACC. NUM. COUNT: 1  
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2003219813	A1	20031127	US 2003-406073	20030403
PRIORITY APPLN. INFO.:			US 2002-369876P	P 20020405

AB The invention relates to SCUBE mols. and generally to gene expression in vascular endothelial cells. The invention specifically relates to the discovery of a novel gene family containing the genes and proteins referred to herein as SCUBE1, SCUBE2 and SCUBE3 which can be expressed in endothelial cells. SCUBE1, SCUBE2 and SCUBE3 genes are found to reside on human chromosome 22q13 and 11p15, and 6p21 resp. SCUBE genes encode secreted proteins containing a secretory signal region, a chain of EGF-like domains, and a CUB domain, that can be differentially expressed in human endothelial cells compared to other human cell types. For an example, SCUBE1 contains signal peptide (amino acids 1-22), 10 EGF-like domains (amino acids 37-72; 78-115; 121-156; 166-202; 206-241; 245-280; 286-321; 327-360; 366-401; and 737-773 resp.), a spacer region (amino acid 487-503) and a CUB domain (amino acids 798-907). For the characterization of SCUBE1 function, provided are recombinant proteins of two SCUBE1 deletion proteins, containing amino acids 26-789 (deleting the CUB domain) and 26-411 (deleting the spacer, the 10th EGF-like domain and the CUB domain) resp. are prepared. The spacer region is critical for the secretion and surface expression of SCUBE1. SCUBE1 is also a glycosylated membrane associated protein with homomeric interaction. Down-regulation of SCUBE1 and SCUBE2 expression by cellular signaling factors (IL-1 $\beta$ , TNF- $\alpha$ ) or by Toxins (LPS) are demonstrated in monkey tissues. Furthermore, SCUBE1 is shown to interact with growth factors PDGF-C or PDGF-D.

. SCUBE proteins may be involved in the development of cardiovascular disease, hemostasis, thrombosis, inflammatory disease, bone metabolism disorders, urinary bladder disorders and breast disorders. The expression profiles for SCUBE1, SCUBE2 and SCUBE3 in human, mouse and monkey are provided; and SCUBE1 is detected in thrombi in many monkey tissues, including in kidney and spleen.

L4 ANSWER 4 OF 10 CAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2003:892428 CAPLUS  
DOCUMENT NUMBER: 139:359243  
TITLE: Compositions and methods for modulating vasculogenesis or angiogenesis with platelet-derived growth factor C (PDGF-C) core protein domain  
INVENTOR(S): Li, Xuri; Eriksson, Ulf; Carmeliet, Peter; Collen, Desire  
PATENT ASSIGNEE(S): Ludwig Institute for Cancer Research, USA  
SOURCE: U.S. Pat. Appl. Publ., 82 pp., Cont.-in-part of U.S. Ser. No. 410,349.  
CODEN: USXXCO  
DOCUMENT TYPE: Patent  
LANGUAGE: English  
FAMILY ACC. NUM. COUNT: 5  
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
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US 2003211994	A1	20031113	US 2002-303997	20021126
US 2004053837	A1	20040318	US 2003-439337	20030516
PRIORITY APPLN. INFO.:			US 1998-102461P	P 19980930
			US 1998-108109P	P 19981112
			US 1998-110749P	P 19981203
			US 1998-113002P	P 19981218
			US 1999-135426P	P 19990521
			US 1999-144022P	P 19990715
			US 1999-410349	A2 19990930
			US 2002-303997	A2 20021126

AB A method for modulating vasculogenesis or angiogenesis using the core domain protein of PDGF-C, a new member of the PDGF/VEGF family of growth factors, or a homodimer or a heterodimer comprising the core domain. Also disclosed are pharmaceutical compns. comprising the core protein, nucleotide sequences encoding the protein, and uses thereof in medical and diagnostic applications.

L4 ANSWER 5 OF 10 MEDLINE on STN DUPLICATE 2  
 ACCESSION NUMBER: 2003398231 MEDLINE  
 DOCUMENT NUMBER: PubMed ID: 12937299  
 TITLE: A fully human monoclonal antibody (CR002) identifies PDGF-D as a novel mediator of mesangioproliferative glomerulonephritis.  
 AUTHOR: Ostendorf Tammo; van Roeyen Claudia R C; Peterson Jeffrey D; Kunter Uta; Eitner Frank; Hamad Avin J; Chan Gerlinde; Jia Xiao-Chi; Macaluso Jennifer; Gazit-Bornstein Gadi; Keyt Bruce A; Lichenstein Henri S; LaRochelle William J; Floege Jurgen  
 CORPORATE SOURCE: Division Nephrology, University of Aachen, Germany.  
 SOURCE: Journal of the American Society of Nephrology : JASN, (2003 Sep) Vol. 14, No. 9, pp. 2237-47. Journal code: 9013836. ISSN: 1046-6673.  
 PUB. COUNTRY: United States  
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)  
 LANGUAGE: English  
 FILE SEGMENT: Priority Journals  
 ENTRY MONTH: 200402  
 ENTRY DATE: Entered STN: 26 Aug 2003  
 Last Updated on STN: 5 Feb 2004  
 Entered Medline: 4 Feb 2004

AB PDGF-B is of central importance in mesangioproliferative diseases. PDGF-D, a new PDGF isoform, like PDGF-B, signals through the PDGF betabeta-receptor. The present study first determined that PDGF-D is mitogenic for rat mesangial cells and is not inhibited by a PDGF-B antagonist. Low levels of PDGF-D mRNA were detected in normal rat glomeruli. After induction of mesangioproliferative nephritis in rats by anti-Thy 1.1 mAb, glomerular PDGF-D mRNA and protein expression increased significantly from days 4 to 9 in comparison with nonnephritic rats. Peak expression of PDGF-D mRNA occurred 2 d later than peak PDGF-B mRNA expression. In addition, PDGF-D serum levels increased significantly in the nephritic animals on day 7. For investigating the functional role of PDGF-D, neutralizing fully human mAb were generated using the XenoMouse technology. Rats with anti-Thy 1.1-induced nephritis were treated on days 3 and 5 with different amounts of a fully human PDGF-DD-specific neutralizing mAb (CR002), equal amounts of irrelevant control mAb, or PBS by intraperitoneal injection. Specific antagonism of PDGF-D led to a dose-dependent (up to 67%) reduction of glomerular cell proliferation. As judged by double immunostaining for 5-bromo-2'-deoxyuridine and alpha-smooth muscle actin, glomerular mesangial cell proliferation was reduced by up to 57%. Reduction of glomerular cell proliferation in the rats that received CR002 was not associated with reduced glomerular expression of PDGF-B mRNA. PDGF-D antagonism also led to reduced glomerular infiltration of monocytes/macrophages (day 5) and reduced accumulation of fibronectin (day 8). In contrast, no effect was noted in normal rats that received an injection of CR002. These data show that PDGF-D is overexpressed in mesangioproliferative states and can act as an auto-, para-, or even endocrine glomerular cell mitogen, indicating that antagonism of PDGF-D may represent a novel therapeutic approach to mesangioproliferative glomerulonephritides.

L4 ANSWER 6 OF 10 CAPLUS COPYRIGHT 2006 ACS on STN  
 ACCESSION NUMBER: 2002:850240 CAPLUS  
 DOCUMENT NUMBER: 137:363702  
 TITLE: Platelet-derived growth factor D, DNA coding for it, and pharmaceutical uses  
 INVENTOR(S): Eriksson, Ulf; Aase, Karin; Li, Xuri; Ponten, Annica; Uutela, Marko; Alitalo, Kari; Oestman, Arne; Heldin,

PATENT ASSIGNEE(S): Carl-Henrik  
 SOURCE: Swed.  
 U.S. Pat. Appl. Publ., 60 pp., Cont.-in-part of U. S.  
 Ser. No. 691,200, abandoned.  
 CODEN: USXXCO  
 DOCUMENT TYPE: Patent  
 LANGUAGE: English  
 FAMILY ACC. NUM. COUNT: 4  
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2002164710	A1	20021107	US 2002-86623	20020304
US 6706687	B1	20040316	US 1999-438046	19991110
US 2003073637	A1	20030417	US 2002-260539	20021001
US 7105481	B2	20060912		
US 2005209136	A1	20050922	US 2004-794392	20040308
US 2006030695	A1	20060209	US 2005-140284	20050531

PRIORITY APPLN. INFO.:

US 1998-107852P	P	19981110
US 1998-113997P	P	19981228
US 1999-150604P	P	19990826
US 1999-157108P	P	19991004
US 1999-157756P	P	19991005
US 1999-438046	A2	19991110
US 2000-691200	B2	20001019
US 2002-86623	A2	20020304
US 2002-260539	A2	20021001

AB PDGF-D, a new member of the PDGF/VEGF family of polypeptide growth factors, is described, as well as nucleotide sequences encoding, methods for producing, pharmaceutical compns. containing this new growth factor, and its antibodies and other antagonists. Also disclosed are transfected and transformed host cells expressing PDGF-D, and uses thereof in medical and diagnostic applications. Fragments and homologs of PDGF-D are also covered by the invention.

L4 ANSWER 7 OF 10 CAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2002:540192 CAPLUS

DOCUMENT NUMBER: 137:104171

TITLE: PDGF D polypeptides, nucleic acids encoding them, and therapeutic or diagnostic applications of the polypeptides or their antibodies

INVENTOR(S): Shimkets, Richard A.; Lichenstein, Henri; Herrmann, John L.; Boldog, Ferenc L.; Minskoff, Stacey; Jeffers, Michael; Andrews, David; La Rochelle, William

PATENT ASSIGNEE(S): USA

SOURCE: U.S. Pat. Appl. Publ., 97 pp., Cont.-in-part of U.S. Ser. No. 715,332.

CODEN: USXXCO

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 168

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2002094546	A1	20020718	US 2001-775482	20010202
WO 2002059618	A2	20020801	WO 2001-US48901	20011116
WO 2002059618	A3	20030508		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH,

PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA,  
 UG, US, UZ, VN, YU, ZA, ZW  
 RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY,  
 KG, KZ, MD, RU, TJ, TM, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB,  
 GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA,  
 GN, GQ, GW, ML, MR, NE, SN, TD, TG

AU 2005200106	A1	20050210	AU 2005-200106	20050112
AU 2006201467	A1	20060504	AU 2006-201467	20060407
PRIORITY APPLN. INFO.:			US 1999-158083P	P 19991007
			US 1999-159231P	P 19991013
			US 2000-174485P	P 20000104
			US 2000-186707P	P 20000303
			US 2000-188250P	P 20000310
			US 2000-223879P	P 20000808
			US 2000-234082P	P 20000920
			US 2000-688312	A2 20001013
			US 2000-715332	A2 20001116
			AU 2000-37360	A3 20000309
			AU 2000-78680	A3 20001006

AB Disclosed are novel PDGFD nucleic acids encoding proteins and polypeptides related to bone morphogenetic protein-1 (BMF1), to vascular endothelial growth factor E (VEGF-E) and to platelet derived growth factor (PDGF). Also disclosed are vectors, host cells, antibodies, and recombinant methods for producing these nucleic acids and polypeptides. Methods of use include detecting and staging of cancers. The claims of this continuation-in-part patent specifically claim a method of detecting the presence of at least one PDGFD antigen in a sample, comprising the steps of: (a) providing a biol. sample; (b) contacting the sample with an agent that binds the antigen; and (c) detecting the presence of the agent bound to the antigen; whereby the presence of the agent indicates that the antigen is present in the sample. A method contributing to a diagnosis of cancer in a subject based on the presence of a PDGFD antigen in a sample from the subject is also claimed, as is a method of staging cancer in a subject. Addnl. claimed are a method of phosphorylating a tyrosine residue of a cellular receptor comprising the step of contacting a cell harboring the receptor with a PDGFD polypeptide, a method of stimulating a response in a cell that is specific for a PDGF beta receptor comprising contacting the cell with a PDGFD polypeptide, and a method of inhibiting the growth of a cell by contacting the cell with an agent that specifically binds a PDGFD polypeptide. An isolated nucleic acid comprising a sequence encoding a PDGFD polypeptide and a method of preparing the PDGFD polypeptide are also claimed.

L4 ANSWER 8 OF 10 CAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2002:409185 CAPLUS

DOCUMENT NUMBER: 137:5012

TITLE: Anti-zvegfg4 antibodies,  
 zvegfg4 antagonists, and antisense  
 polynucleotides for treating fibroproliferative  
 disorders

INVENTOR(S): Hart, Charles E.; Topouzis, Stavros; Gilbertson, Debra  
 G.

PATENT ASSIGNEE(S): USA

SOURCE: U.S. Pat. Appl. Publ., 34 pp., Cont.-in-part of U. S.  
 Ser. No. 564,595.  
 CODEN: USXXCO

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 3

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
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US 2002064832	A1	20020530	US 2001-808972	20010314



US 6630142	B2	20031007		
US 6495668	B1	20021217	US 2000-564595	20000503
US 2004002140	A1	20040101	US 2001-876813	20010606
US 6962802	B2	20051108		
US 2003105015	A1	20030605	US 2002-226559	20020823
US 6866991	B2	20050315		
US 2004043027	A1	20040304	US 2003-606055	20030625
US 2004242850	A1	20041202	US 2004-877623	20040625
US 2005031694	A1	20050210	US 2004-910938	20040803
US 2005164937	A1	20050728	US 2005-80803	20050315

PRIORITY APPLN. INFO.:

US 1999-132250P	P	19990503
US 1999-164463P	P	19991110
US 2000-180169P	P	20000204
US 2000-564595	A2	20000503
US 2000-235295P	P	20000926
US 2000-540224	A3	20000331
US 2001-808972	A3	20010314
US 2001-876813	A3	20010606
US 2002-226559	A1	20020823

AB Materials and methods for reducing cell proliferation or extracellular matrix production in a mammal are disclosed. The methods comprise administering to a mammal a composition comprising a therapeutically effective amount of a zvegf4 protein antagonist in combination with a pharmaceutically acceptable delivery vehicle. Exemplary zvegf4 antagonists include anti-zvegf4 antibodies, inhibitory polynucleotides, inhibitors of zvegf4 activation, and mitogenically inactive, receptor-binding variants of zvegf4. The materials and methods are useful in the treatment of, inter alia, fibroproliferative disorders of the kidney, liver, and bone.

L4 ANSWER 9 OF 10 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN DUPLICATE 3

ACCESSION NUMBER: 2002162477 EMBASE  
 TITLE: Platelet-derived growth factor D: Tumorigenicity in mice and dysregulated expression in human cancer.  
 AUTHOR: LaRoche W.J.; Jeffers M.; Corvalan J.R.F.; Jia X.-C.; Feng X.; Vanegas S.; Vickroy J.D.; Yang X.-D.; Chen F.; Gazit G.; Mayotte J.; Macaluso J.; Rittman B.; Wu F.; Dhanabal M.; Herrmann J.; Lichenstein H.S.  
 CORPORATE SOURCE: W.J. LaRoche, 22 East Main Street, Branford, CT 06405, United States. wlarochelle@curagen.com  
 SOURCE: Cancer Research, (1 May 2002) Vol. 62, No. 9, pp. 2468-2473. .  
 Refs: 22  
 ISSN: 0008-5472 CODEN: CNREA8  
 COUNTRY: United States  
 DOCUMENT TYPE: Journal; Article  
 FILE SEGMENT: 016 Cancer  
 LANGUAGE: English  
 SUMMARY LANGUAGE: English  
 ENTRY DATE: Entered STN: 16 May 2002  
 Last Updated on STN: 16 May 2002

AB Platelet-derived growth factor (PDGF) has been directly implicated in developmental and physiological processes, as well as in human cancer and other proliferative disorders. We have recently isolated and characterized a novel protease-activated member of the PDGF family, PDGF D. PDGF D has been shown to be proliferative for cells of mesenchymal origin, signaling through PDGF receptors. Comprehensive and systematic PDGF D transcript analysis revealed expression in many cell lines derived from ovarian, renal, and lung cancers, as well as from astrocytomas and medulloblastomas.  $\beta$  PDGF receptor profiling further suggested autocrine signaling in several brain tumor cell lines. PDGF D transforming ability and tumor formation in SCID mice was

further demonstrated. Exploiting a sensitive PDGF D sandwich ELISA using fully human monoclonal antibodies, PDGF D was detected at elevated levels in the sera of ovarian, renal, lung, and brain cancer patients. Immunohistochemical analysis confirmed PDGF D localization to ovarian and lung tumor tissues. Together, these data demonstrate that PDGF D plays a role in certain human cancers.

L4 ANSWER 10 OF 10 MEDLINE on STN DUPLICATE 4  
 ACCESSION NUMBER: 2002667552 MEDLINE  
 DOCUMENT NUMBER: PubMed ID: 12427128  
 TITLE: Platelet-derived growth factor-D expression in developing and mature human kidneys.  
 AUTHOR: Changsirikulchai Siribha; Hudkins Kelly L; Goodpaster Tracy A; Volpone John; Topouzis Stavros; Gilbertson Debra G; Alpers Charles E  
 CORPORATE SOURCE: Department of Medicine, Srinakharinwirot University, Bangkok, Thailand.  
 CONTRACT NUMBER: DK47959 (NIDDK)  
 SOURCE: Kidney international, (2002 Dec) Vol. 62, No. 6, pp. 2043-54.  
 Journal code: 0323470. ISSN: 0085-2538.  
 PUB. COUNTRY: United States  
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)  
 LANGUAGE: English  
 FILE SEGMENT: Priority Journals  
 OTHER SOURCE: GENBANK-AF336376  
 ENTRY MONTH: 200305  
 ENTRY DATE: Entered STN: 13 Nov 2002  
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AB BACKGROUND: Platelet-derived growth factor (PDGF) is a family of growth regulatory molecules composed of sulfide-bonded dimeric structures. Two well-studied PDGF peptides (PDGF-A and PDGF-B) have been shown to mediate a wide range of biological effects. PDGF-D is a newly recognized member of the PDGF family. Initial studies of the PDGF -D gene found its expression in cells of the vascular wall, suggesting that it could participate in vascular development and pathology. However, its localization in human kidney tissues has never been studied. METHODS: PDGF-D expression in fetal (N = 30) and adult (N = 25) human kidney tissues was examined by immunohistochemistry using an affinity-purified antibody raised to human PDGF-D. Antibody absorption with the immunizing peptide was employed to confirm the specificity of this antibody. PDGF-D protein and gene expression in human kidneys also were demonstrated by Western blotting and reverse transcription-polymerase chain reaction (RT-PCR). RESULTS: In the developing kidney, PDGF-D was first expressed by epithelial cells of comma- and S-shaped structures of the developing nephron, and most consistently in the visceral epithelial cells in the later stages of glomerular differentiation. In addition, PDGF-D could be found in mesenchymal, presumptively fibroblast cells in the interstitium of developing renal pelvis and in fetal smooth muscle cells in arterial vessels. In the adult normal kidney, PDGF-D was expressed by the visceral epithelial cells. There was persistent expression in arterial smooth muscle cells as well as in some neointimal smooth muscle cells of arteriosclerotic vessels, and expression in smooth muscle cells of vasa rectae in the medulla. PDGF-D could be identified at the basolateral membrane of some injured tubules in areas of chronic tubulointerstitial injury routinely encountered in aging kidneys. Western blotting of homogenates of adult kidneys demonstrated monospecific bands at 50 kD corresponding to previously established size parameter for this protein. RT-PCR of human

kidney RNA resulted in a 918 basepair band, the sequence of which corresponded to human PDGF-D (Genbank number AF336376). CONCLUSIONS: To our knowledge, these are the first studies to localize PDGF-D in human kidneys and suggest that PDGF-D may have a role in kidney development. PDGF-D was shown to bind to PDGF beta receptor, which localizes to mesangial cells, parietal epithelial cells, and interstitial fibroblasts, suggesting potential paracrine interactions between those cells and the visceral epithelium.